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dr Chetty's Covid Treatment Part 3

23-30 minutes

Translation of the original of an article by Dr. Shankara Chetty on his observations and reflections that led to his extremely successful treatment of Covid, which completely avoided deaths and hospitalizations

The article was published in issue 5, August-September 2020, of the South African medical journal *Modern Medicine* under the title **Elucidating the Pathogenesis and Rx of COVID Reveals a Missing Element** .

The text linked as the author's original was translated at : https://8days.org/videos-and-articles

The screenshots of the original can be read in full resolution by clicking on them :

pdf file of the German translation by Dr. Chetty's one-page recommendation for Covid treatment from day 8

It goes without saying that this is all information about how Dr. Shankara Chetty treated for Covid. From the following translation and also from the interviews with Dr. Shankara Chetty, whose translation I am working on, makes it clear that unfortunately his method is NOT suitable for self-treatment, it requires a doctor to do it. Seen in this way, my translations and articles in the Shankara Chetty category are only intended as suggestions for doctors and health politicians, but also for everyone who is looking for an ultimate argument against vaccination and against compulsory vaccination.

The method of dr. Chetty works with approved medications and can be learned and used by any general practitioner in a short time. dr Chetty also mentions in his latest interview from December 4th that he very successfully trained first leading doctors and then rural doctors in a province of India to use his method via distance learning. If the federal and state governments are really concerned about the health of the population, as they repeatedly claim, then this is a great opportunity for our politicians, which can be implemented immediately, to refute conspiracy theories and to prove that they can do this with health really serious.

start of translation

Elucidating the pathogenesis and prescription for treating COVID brings to light a missing element

SUMMARY

I am a Scientific Biologist trained in Genetics, Advanced Biology, Microbiology and Biochemistry and a General Practitioner currently in private practice in Port Edward, South Africa. This article is based on my observation and treatment of the first 200 patients with Covid disease from the first wave of the current pandemic. Written in July 2020, it was widely circulated and subsequently published in Modern Medicine, an academic peer-reviewed journal in South Africa.

In essence, I have found that Covid disease is a biphasic, non-linear disease with little correlation between the two phases. The first phase consists of a typical, self-limiting viral disease of varying severity affecting the respiratory and/or gastrointestinal tract. Most patients show signs of improvement by day 6 after the onset of symptoms. The second phase consists of an inflammatory process that occurs in a proportion of these patients and begins exactly on the eighth day after the onset of symptoms (a week later). This critical second process is triggered by a hypersensitivity reaction of varying severity and, if not treated in time, leads to hypercoagulability via hyperinflammation.

I have currently treated over 6500 patients with this perspective, spanning three waves of this pandemic, always with the same result: no deaths or hospitalizations, and I have largely negated the need for appreciable oxygen demand by reversing hypoxia in a timely manner.

INTRODUCTION

Currently, there are few recommendations for outpatient treatment and there is a distinct lack of knowledge about the course of the disease. This is most likely due to insufficient outpatient assessment, treatment and follow-up due to isolation measures and current protocols. However, there is a wealth of information about hospitalizations, examinations and pathological findings. The treatment protocols in the hospitals are based on these findings, but so far have not been consistently consistent in terms of effectiveness and outcomes. This has led to controversy and confusion regarding the pathogenesis and treatment of COVID.

Over the past five months, my staff and I have evaluated, treated, and followed up more than 200 symptomatic COVID patients, some of whom were critically ill. In doing so, I used the information gathered to refine my understanding of the pathogenesis of COVID and thus adapt treatment protocols.

This has led to some remarkable, but consistent and predictable, results and recoveries. Overall, we had no deaths, no hospitalizations, and full recovery for all patients, even those with severe dyspnea. Most corroborating my theory of pathogenesis were the 12 most severely dyspneic patients with low SpO2 of 80% who recovered to over 96% SpO2 within 24 to 36 hours of treatment without the need for hospitalization or oxygen. All dyspnea patients had normal SpO2 within 3 days of treatment.

The information gathered in this way can prevent much of the mortality and morbidity caused by COVID. A better understanding of the pathogenesis of COVID can guide future research and intervention strategies to mitigate the impact of the pandemic.

Virus, detection and symptoms

An RNA virus

Transmission through the air. Common in stool samples. Waterborne Transmission. The virus is highly contagious, but infectivity and virulence are unknown due to a lack of knowledge about the pathogenesis of COVID and limited testing capabilities.

The virus enters the cell via ACE-2 receptors. Like other common RNA viruses, it uses cellular machinery to replicate and eject copies, leaving behind dead cellular debris and inflammation that can result in mild scarring. The average duration of symptoms is 3 to 6 days, with the host no longer infectious by day 7.

Detection in the laboratory

The ability to isolate the virus varies greatly between swabs and depends on the technique used, the training of the inspectors, the area swabbed, etc. PCR tests are very specific but not very sensitive - about 65%, ie about 35% false negative results - and therefore cannot be used to diagnose or confirm the diagnosis. It is only useful for screening purposes, but not sensitive enough to drive detection and isolation/quarantine measures. Absolute numerical values are not meaningful; Ratios can provide better insight Antibody tests can provide more reliable data.

Clinical presentation based on personal observations

• Upper respiratory tract infection - sore throat, loss of smell, loss of sweet and salty taste, bitterness retained. Generalized body aches, fever with chills.

• Spreads down - dry persistent cough, coldness between shoulder blades, burning sensation in chest, tightness with scant, clear sputum.

• Bacterial co-infection - productive cough with purulent sputum, sinusitis with purulent mucus, earache, etc. The above symptoms are progressive in the first 6 days of infection and can lead to pneumonia with accompanying dyspnea (shortness of breath).

• A significant proportion of infected symptomatic individuals develop dyspnea by day 7, regardless of the severity or duration of the initial symptoms. Frequent accompanying symptoms are light, generalized body pains and tiredness up to the compulsion to sleep. This dyspnea can occur suddenly and progress rapidly, leading to severe hypoxia and a drop in SpO2 below 85% within 2 days. It usually begins insidiously and lasts for varying lengths of time, with SpO2 in the mid to low 90s, and can progress to diffuse pulmonary fibrosis the longer it persists. Rashes, neurological symptoms, and end-organ damage are also reported at this stage.

• Gastrointestinal infections are common - usually preceded by a sore throat that resolves spontaneously in a day or two, heartburn, nausea, brief, severe, intermittent abdominal cramps with tingling and gurgling, severe diarrhea that resolves after four to five Days turned into a poorly formed, sometimes slimy, stool.

• Other symptoms reported: conjunctivitis, various skin rashes, distal ischemic finger injuries, various neurological symptoms, symptoms of organ damage or failure.

case morphology

To understand the course of this disease, one must also review the facts as they currently stand versus the facts as they are known.

Known facts

Viruses tend to be quite specific in terms of the type of tissue they infect. Their infections are generally self-limiting, opportunistic, and rarely fatal. Mortality is usually due to some other predisposition, either natural or chronic disease.

Respiratory viruses cause symptoms ranging from none (in most cases) to a mild sore throat, which resolves after a few days or spreads deeper and can lead to a bacterial infection ranging from mild bronchitis to pneumonia, with typical radiological findings. These symptoms are progressive and well-researched, and these typical case reports should be removed from the analysis to focus on what is unknown.

facts as they occur

What remains are case histories that do not fit the above profile, are atypical for a single virus, and do not show typical disease courses and rates.

Unusual Symptoms:

• Hypoxia hardly correlates with the degree of dyspnea. Sudden, rapidly progressive dyspnea and a drop in SpO2 in an otherwise healthy patient, leading to a poor outcome.

• Slow chronic hypoxia with variable chronic lung damage from fibrosis over a variable duration. Associated with a persistent, dry cough with or without wheezing. – Slight drop in SpO2, not less than 92%, intermittent oxygen may be required. Usually resolves spontaneously within a few days to a week.

• Autopsy finding: The lungs are edematous and covered with microvascular clots. Multiorgan involvement is usually due to hypoxic injury, DIC, or an immune/inflammatory response rather than direct viral infection.

• Chronic manifestations: COPD, Kawasaki-like disease in children, hypoxic injuries, thromboembolic injuries, diabetes.

Unusual results:

If we exclude the usual risk factors that can complicate a typical viral infection, what remains is a poor correlation between age and health status. Fit, healthy 25-year-olds have died suddenly, and high-risk 90-year-olds have come through it with no problem. Patients with a mild, long-term illness may come back in a few months with a chronic illness, usually COPD and diabetes. Men are at higher risk of multiple deaths within the family due to an increased risk of infection due to lockdowns and/or genetic predisposition. There are many deaths between father and son where the mother is spared and vice versa, but this is less common. Different mortality rates between countries and ethnic groups. Children under the age of 10 are least at risk.

Pathogenesis by morphology

From the morphology of the cases, it is clear that viral infection alone cannot explain the variety of symptoms, unusual presentations, and unusual outcomes. An overview of the wealth of information that is available about the pathogenesis of a variety of diseases may allow us to find the best solution to the unusual presentations and outcomes that are observed.

The only pathogenesis that fully explains these results is type 1 hypersensitivity reactions, that is, allergic reactions to external allergens, whether inhaled, ingested, or touched. These reactions consist of an initial acute phase lasting a few hours to a few days and can be mild to fatal. Sometimes there is a delayed reaction that lasts about a week and leads to cell damage and other effects on the immune system. Reactions to the same allergen vary in speed, severity, duration and symptoms and, if left untreated, have varied outcomes ranging from sudden anaphylactic reactions leading to rapid deterioration in quality of life and death,

In my opinion and based on my study, treatment and review of over 200 COVID patients, the initial infection with Corona virus is like any other common respiratory virus infection, with a similar statistical distribution as in previous epidemics, during the first 7 days. A type 1 hypersensitivity reaction is elicited in the lungs around day seven, likely due to a recognizable, allergenic viral protein fragment causing release of chemical mediators, leading to the various manifestations and outcomes observed to date, including chronicity and complications due to non-treatment. This reaction would not be directly related to age, comorbidities, etc.,

This could explain the sudden deterioration in lung oxygen exchange capacity and SpO2 in an asymptomatic and mild transient viral disease around the seventh day. The rate of deterioration varies widely and can complicate an otherwise uneventful recovery in a high-risk patient after day 7.

In severe type 1 reactions with sudden onset of dyspnea and steadily decreasing SpO2, the situation can deteriorate rapidly and there is a high risk of death. However, some individuals with milder initial reactions that progress to late-stage type 1 hypersensitivity reactions present with persistent dry cough, symptoms of mild hypoxia or hypoxic injury, etc., with a slight but sustained drop in SpO2. These have varying degrees of lung damage over time.

Many of the reported chronic manifestations of COVID are explained by immune damage to the lungs (cytokine response) and collateral immune or hypoxic damage to other organs or systems. The gastrointestinal symptoms are probably due to an initial viral gastroenteritis, followed by a long-lasting allergic intestinal inflammation and irritability with chronic sequelae.

BCG (tuberculosis) vaccination and active PTB (pulmonary tuberculosis) vaccination appear to modulate immunity and prevent severe type 1 reactions. Patients treated with immunomodulators are less likely to develop a severe type 1 reaction.

Children's underdeveloped immunity is less likely to result in a type 1 reaction. Generally, younger patients do not show any reactions as they are being exposed to the allergen for the first time. A reaction requires prior exposure. However, they become sensitized, and further exposure may provoke a stronger immune response. Those who initially show mild to moderate reactions become more tolerant with later exposures. However, this makes them passive future carriers of the virus.

As reports of reinfection emerge, a Type 1 response could explain a longer second wave of infection with higher mortality in the younger population (sensitized individuals) and a shorter third wave with generally low mortality (tolerance) as in Spanish flu.

Tool box for treatment

Since I believe this disease has two overlapping etiologies, one viral and one allergic, treatment would differ depending on when it is initiated.

Drugs used on an outpatient basis

Hydroxychloroquine is very controversial and has long been used prophylactically against viral infections; in studies of health care workers it has shown some prophylactic benefit. It has anti-inflammatory, antihistamine, smooth muscle relaxant and antiarrhythmic properties. This could have symptomatic benefit during the viral phase of COVID disease. Its immunomodulatory effects would be of greater benefit in the allergic response but may have too slow an onset to be of benefit if medication is started later in the first 7 days. Ivermectin's immunomodulatory effect may have a faster onset and its ability to clear pulmonary eosinophilia may be beneficial.

• Azithromycin has been shown to be beneficial in the treatment of common and atypical bronchopneumonia complicating viral infections and should be the antibiotic of choice in cases complicated by bacterial URTIs.

• Doxycycline has a wide range of effects and may slow viral replication through its inhibitory effect on protein synthesis. This can potentially reduce the severity of symptoms and the contagiousness of those infected.

• The viral phase of the disease is generally mild and self-limiting, so symptomatic treatment is sufficient in most cases.

Drugs used to treat type 1 hypersensitivity reactions

• Adrenaline is used to treat hypovolemic shock. It can also be used by nebulization to inhalation in patients with rapidly progressive reactions and severe dyspnea.

• Prednisone is indicated to suppress a sudden severe allergic reaction. Its application from the 7th day can be life-saving. Intake within the first 7 days can be harmful and must be limited to life-threatening illnesses during this period.

• Promethazine is the antihistamine of choice for Type 1 hypersensitivity reactions. It can quickly and effectively suppress all immediate manifestations of Type 1 reactions. Gastrointestinal symptoms may require the use of H2 antagonists.

• Montelukast, a leukotriene receptor antagonist, blocks the action of cysteinyl leukotrienes, a unique property unmatched by corticosteroids. It has both bronchodilator and anti-inflammatory effects. It is indicated for the prophylaxis and treatment of atopic diseases and has utility in the prevention and treatment of type 1 reactions.

• Beclomethasone is an inhaled steroid that can topically suppress pneumonia. It would be beneficial in patients with prolonged reactions and associated dry cough. It could also limit pulmonary fibrosis and progression of COPD.

Other less common drugs that should be of use are: ipratropium bromide / sodium chromoglycates / ketotifen.

protocol

viral phase

All patients should be asked about the day of onset of illness and clearly documented.

Mild symptoms: sore throat, loss of smell, etc.

• Hydroxychloroquine 200mg daily x 5 days

• Montelukast 10mg daily x 1 month

• Treat symptomatically

Moderate symptoms: dry cough, mucopurulent bronchitis, etc.

• Hydroxychloroquine 200mg daily x 5 days

• Azithromycin 500mg on day 1, then 250mg daily for 4 more days, or another more appropriate antibiotic

• Montelukast 10mg daily x 1 month

• Treat symptomatically

Most patients recover quickly from mild symptoms. Those with moderate symptoms take a little longer.

All patients should be advised to be alert for new symptoms after day 7, even if fully recovered, and to report for treatment immediately. These symptoms are usually: general body aches, fatigue, dyspnea, and/or decreasing SpO2. These herald the onset of the hypersensitivity reaction.

phase of hypersensitivity

Range of presentations: rapidly progressive dyspnea with SpO2 below 80% with or without chest symptoms to slow, sustained SpO2 decline, persistent coughing, wheezing, etc.

• Prednisone 50mg stat and decrease the e once daily, morning dose by 5mg over the next 9 days...50, 45, 40, 35mg mane... Those presenting with mild, persistent symptoms may need lower doses, those over a longer period period can be reduced.

• Promethazine 25mg stat (immediately) then tds (3x daily) x 5 days.

• Adrenaline, nebulized, inhale for severe dyspnea or suspected hypotension

• Aspirin prophylaxis, morning x 1 month.

• Montelukast 10 mg nocte (at night, before bedtime) x 1 month.

• Naproxen 250 mg bd (twice a day) for fever because it is an allergic inflammation and not an infection. Paracetamol is not effective.

• Beclate 200mcg inhaler bd (2x daily) for people with chronic dry cough (topical steroid)

• Sodium chromoglycate/ketotifen/ipratropium bromide inhalers may provide better results and possible prophylactic benefit

future infections

Patients who do not develop a hypersensitivity reaction during initial infection either have not been previously exposed or are tolerant. Specific IgE screening would identify those at risk for subsequent reactions, and significantly elevated IgE would identify those prone to severe reactions. Montelukast would prevent these reactions and should be used prophylactically in individuals with elevated IgE levels.

observations

The following is based on my personal observations of studying over 200 COVID patients from presentation to full recovery using the above treatment protocol. Numerous observations confirm the presence of a type 1 hypersensitivity reaction.

Hydroxychloroquine and Doxycycline

Early initiation of hydroxychloroquine helps symptomatically and may suppress the hypersensitivity reaction by day 7. However, it is less effective than other drugs at affecting immune hypersensitivity when used later in the disease.

Doxycycline has been used prophylactically in a large group (160) of high-risk individuals (teachers and police officers) in the past three months. So far, fewer people have been infected in the prophylaxis group than in their colleagues' group.

The four people who became infected showed no to mild transient symptoms that resolved spontaneously during the viral phase. They have been isolating at home and none of their close contacts have tested positive or had any symptoms throughout their illness. This could be an indication of the suppressive effect of doxycycline on viral replication and consequently on viral transmission. However, three of them developed dyspnoea on day 7, which improved rapidly with treatment. The assessment is not yet complete.

All other drugs were used depending on the bacterial infection and the symptoms presented, with their usefulness being fairly obvious.

Patients who experienced dyspnea or decreased SpO2 after day 7 were treated immediately as described. All had improved symptoms and SpO2 within 24 hours. Most revealing was a group of the 12 most hypoxic patients, all presenting after day 7, with SpO2 below 80%, all with severe dyspnoea, etc. All had symptom relief within a few hours and SpO2 Value rose again to over 96% within 24 to 36 hours after the start of treatment. This was achieved through outpatient treatment with room air without oxygen, and all 12 made full recoveries within a few days.

montelukast, prednisone and promethazine

Most patients treated with montelukast for the first 7 days had no response by day 7 or thereafter. (About 80 symptomatic patients)

Promethazine effectively eliminated chemical mediators, preventing damage to the lungs and the consequent release of cytokines, resulting in rapid relief from dyspnea. His ability to reverse hypoxia in time is unparalleled.

Prednisone, promethazine and montelukast have been shown to be life-saving and, having treated over 200 COVID patients, we have yet to record a single death or hospitalization. All made a full recovery within 14 days of onset.

No other drug currently used to treat COVID 19 (remdesivir, tocilizumab, convalescent plasma, etc.) has demonstrated such rapid response and predictable success in critically ill patients, requiring no oxygen or hospitalization.

Impact of Observations

The rapid response to the drugs used to treat type 1 hypersensitivity reactions confirms their existence. This could have serious consequences for the future handling of the COVID pandemic.

Screening for a hypersensitivity reaction and prompt treatment would significantly reduce morbidity and mortality. Those who initially have mild to moderate disease develop tolerance with later exposure. However, those who were initially asymptomatic because they were exposed for the first time become sensitized and are at risk of later reactions.

Identifying the specific IgE involved in this response and quantifying its levels would help identify the risk groups. This would also help predict the severity of the response to future exposure and guide prophylactic and preventative treatment.

Vaccines against the virus would only benefit those who are hypersensitive, and widespread vaccination would be unnecessary and unsafe given the rush to market without long-term evaluation. Being able to identify hypersensitive individuals and provide them with appropriate information and treatment can eliminate the need for a vaccine altogether.

conclusion

Given the high mortality and morbidity associated with COVID 19, I hope that the information presented here will help save lives and support further research and treatment. The protocol and its shortcomings provide a valuable starting point for further evaluation of treatment interventions. I hope this brings some clarity at this difficult time.

dr Shankara Chetty.

end of translation

Overview of all 7 parts of this series with links:

- dr Chetty's Covid Treatment Part 1 Translation of Dr. Chetty's discovery and method for treating Covid in Modern Medicine, August-September 2020.
- dr Chetty's Covid Treatment Part 2 Translation of a webinar lecture by Dr. Chetty, Aug. 21, 2021 at Covexit.com
- dr Chetty 's Covid Treatment Part 3 Chetty in Modern Medicine, August-September 2020. Dr. Chetty described his observations and his method after 200 successful Covid treatments at the time.
- dr Chetty's Covid Treatment Part 4 Interview by Dr. Mobeen Sayed with Dr. Chetty, probably early Nov. 2021
- dr Chetty's Covid Treatment Part 5 Link to the highly recommended Corona Committee simultaneous translation interview on 10 Dec 2021, with Dr. chetty
- dr Chetty's Covid Treatment Part 6 Interview by Dr. Philip McMillan with Dr. Shankara Chetty on Dec 4, 2021
- dr Chetty's Covid Treatment Part 7 Translation of an interview by Jean-Pierre Kiekens with Dr. Chetty, 22 Dec 2021 at Covexit.com
- Translation of the abstract by Dr. Chetty's Phase 2 Treatment Protocol . As a pdf file. For the information of doctors only.